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Posterior Fast Atrioventricular Node Pathways: Implications for Radiofrequency Catheter Ablation of Atrioventricular Node Reentrant Tachycardia

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Objectives. **This study sought to present evidence that fast atrioventricular (AV) node pathways with posterior exit sites may participate in typical AV node reentry.**

Background. **Catheter ablation of the slow AV node pathway in the posteroseptal right atrium is the preferred therapeutic approach in patients with AV node reentrant tachycardia. Despite the success achieved with this approach, electrophysiologic changes consistent with fast pathway ablation are occasionally observed. One potential explanation is the presence of an aberrant posterior fast pathway.**

Methods. **The location of fast and slow AV node pathways was determined by atrial activation mapping along the tricuspid valve annulus during tachycardia and was further confirmed by the effect of radiofrequency catheter ablation.**

Results. **Seven patients with AV node reentrant tachycardia had evidence of a posterior fast pathway near the coronary sinus os. Abolition of anterograde and retrograde fast pathway conduction followed radiofrequency ablation in the posteroseptal region in six patients. Consistent with fast pathway ablation, the AH interval increased from 70 ± 24 to 195 ± 35 ms (mean ± SD), and tachycardia was no longer inducible. Selective slow pathway ablation was performed in one other patient with a posterior fast pathway.**

Conclusions. **Functionally fast AV node pathways may be located in the posteroseptal right atrium, where slow pathway modification is performed. These data delineate the limitations of an anatomically guided slow pathway ablative approach and emphasize the importance of detailed mapping and localization of the retrograde fast pathway exit site before ablation. Failure to recognize the presence of posterior fast AV node pathways may account for sporadic examples of AV block, complicating posteroseptal ablation in patients with AV node reentry.**

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Current understanding of atrioventricular (AV) node reentrant tachycardia is based on a model of dual AV node pathways that are functionally and anatomically distinct, with a fast pathway located anterior to the compact AV node in the region of an anterior atrionodal input and a slow pathway located in the posteroseptal right atrium in the region of a posterior atrionodal input (1–4). Based on this spatial segregation of the fast and slow pathways, percutaneous catheter ablation techniques have been developed to selectively abolish or modify fast or slow AV node pathway conduction (1,2,5–13). Although selective slow pathway ablation is performed posterior to the compact AV node, changes in residual AV node conduction consistent with selective fast pathway ablation have been observed in up to 12% of patients undergoing AV node modification using a posterior approach (8,10,11).

In addition to direct damage to the compact AV node, there are at least two other potential explanations for impaired AV node function after posteriorly directed ablation. 1) Selective slow pathway ablation may eliminate or modify conduction of the posterior input to the AV node. Atrioventricular node conduction is thought to depend on summation of functionally distinct dual atrionodal inputs (anterior and posterior), with faster conduction but a lower safety factor for impulse propagation proceeding over the fast anterior input. Elimination of the posterior input (slow conduction but higher safety factor for conduction) can thus result in prolongation of the Wenckebach cycle length and AV node refractory period and high degree AV block (4). 2) An alternative explanation for prolonged AV nodal conduction after radiofrequency catheter ablation in the posteroseptal region is direct damage to an atypically located fast AV node pathway that is located posterior and inferior to the compact AV node. The purpose of the present study is to present evidence to support this latter hypothesis.

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Methods

Electrophysiologic study. Seven of 130 consecutive patients referred for radiofrequency catheter ablation of AV node reentrant tachycardia had evidence of a posterior fast pathway. Atrioventricular node reentry was defined using standard electrophysiologic criteria (14,15). All patients were studied in the postabsorptive state after informed consent had been obtained. Antiarrhythmic agents were discontinued for at least 5 half-lives before the study. Patients received local anesthesia with 0.25% bupivacaine and sedation with intravenous midazolam before electrophysiologic study. Three quadripolar catheters and one decapolar catheter were introduced percutaneously and positioned at the high right atrium, His bundle, right ventricular apex and within the coronary sinus under fluoroscopic guidance. A quadripolar catheter with a 4-mm deflectable tip was used to map atrial activity along the tricuspid valve annulus. Bipolar intracardiac electrograms were filtered at 40 to 400 Hz and displayed simultaneously with three surface electrocardiographic leads (I, aVF, V_1) on a multichannel oscilloscope. Real-time recordings were made with a paper recorder (Astro-Med Inc.) at recording speeds of 50 to 200 mm/s, and data were recorded on magnetic tape. Peripheral arterial pressure was continuously monitored (Dinamap, Critikon Inc.). Programmed stimulation was performed with a programmable stimulator and an isolated constant-current source (Bloom Associates). Stimuli were delivered as rectangular pulses of 2 ms duration at four times diastolic threshold. The stimulation protocol included rapid atrial and ventricular pacing at multiple cycle lengths and the introduction of single, double and triple extrastimuli from atrial and ventricular sites at multiple paced cycle lengths. Atrioventricular node conduction and refractory periods were determined before and 30 min after successful radiofrequency application.

Retrograde AV node pathways were classified as fast (HA interval <100 ms), intermediate (100 to 200 ms) or slow (>200 ms) (16). The location of the fast AV node pathway was determined by identifying the site of earliest atrial activation during the slow–fast form of AV node reentrant tachycardia and was classified as anterior or posterior, depending on the temporal relationship of atrial activation recorded by the His bundle and coronary sinus os catheters. To differentiate the atrial and ventricular electrograms during tachycardia, atrial and ventricular activation were advanced with extrastimuli during tachycardia. A retrograde fast pathway was considered to exit posteriorly if retrograde atrial activation in the coronary sinus os 1) preceded His bundle atrial activation, or 2) was recorded simultaneously in the region of the coronary sinus os and His bundle and posteroseptal radiofrequency ablation resulted in abolition of fast pathway function with associated prolongation of the AH interval.

Radiofrequency ablation. Radiofrequency energy was delivered (10 to 30 W for up to 60 s or until there was an abrupt rise in catheter impedance) from a radiofrequency generator (model RFG-3C RF Lesion Generator, Radionics Inc.). Energy was delivered from the distal 4-mm electrode of the mapping catheter to a posterior chest wall patch (18.4 × 3.8 cm) positioned in the infrascapular region. In all patients slow pathway modification was attempted using a posterior approach (8) with the ablation catheter positioned to record an atrial to ventricular electrogram ratio ≤0.5. For all patients, the tip of the ablation catheter was ≥15 mm posterior to the His bundle electrode, and in no case was a His bundle electrogram recorded from the ablation catheter. Successful ablation was defined by either abolition of fast or slow pathway conduction or induction of a maximum of one AV node echo beat during infusion of isoproterenol (17).

Follow-up evaluation. After the procedure, patients were monitored continuously for 24 h. Follow-up electrophysiologic study was performed in all but two patients (both had high degree AV block) within 48 h after the ablation procedure. During the follow-up study, the success of the ablative procedure was reconfirmed as outlined before, and conduction and refractory properties of the AV node were reevaluated.

Control group. To identify unique characteristics of patients with a posterior fast pathway, we compared the clinical and electrophysiologic variables of the study group with a control group of 40 consecutive patients with AV node reentry and an anterior fast pathway.

Statistics. All data are expressed as mean value ± SD. Comparative data were analyzed by the Mann-Whitney test (a nonparametric analogue of the unpaired t test). The null hypothesis was rejected for $p < 0.05$.

Results

Electrophysiologic findings. There was evidence for a posterior fast AV node pathway in seven patients (52 ± 25 years). In six of seven patients catheter ablation using a posterior approach resulted in changes in residual AV node conduction consistent with anterograde and retrograde fast pathway ablation (Fig. 1). The clinical characteristics of these patients and the results of the electrophysiologic studies are summarized in Table 1.

Six of the seven patients had no evidence for structural heart disease; one patient had coronary artery disease. Dual anterograde AV node pathway physiology was demonstrated in all seven patients, none of whom had discontinuous retrograde AV node conduction curves. All patients had the typical (slow–fast) form of AV node reentrant tachycardia with an induced tachycardia cycle length of 342 ± 68 ms. The AH interval during tachycardia was 289 ± 70 ms, and the VA interval (recorded by the His bundle catheter) was 0 to 25 ms. An example of tachycardia using a posterior fast pathway is shown in Figure 2 (Patient 3). Note that atrial activation at the coronary sinus os precedes atrial activation at the His bundle by 20 ms. In five other patients simultaneous activation of atrial activity was recorded at the coronary sinus os and His bundle during tachycardia. These electrograms were interpreted as being consistent with a fast pathway exit site between the coronary sinus os and His bundle. In the remaining patient

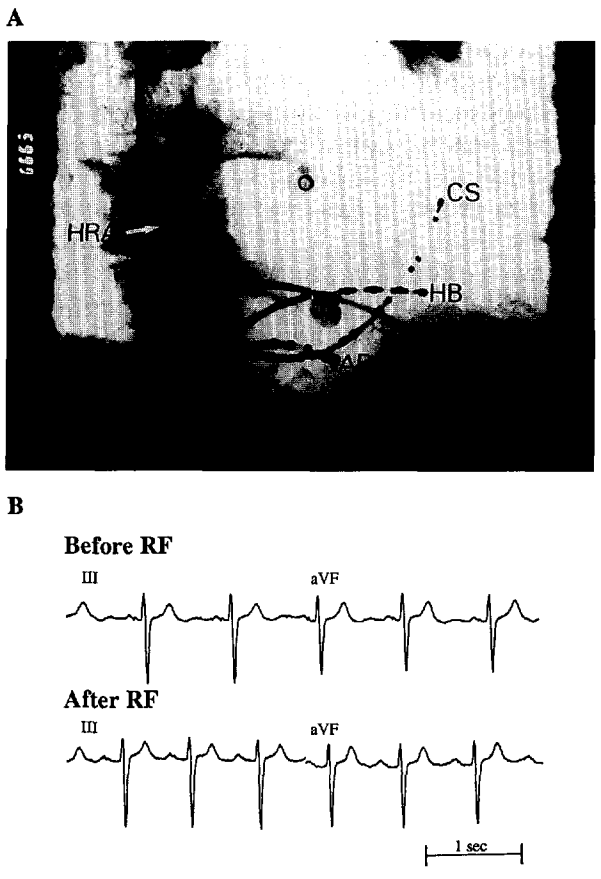


Figure 1. Posterior ablation of the fast atrioventricular (AV) node pathway in a patient with AV node reentrant tachycardia (Patient 2). **A,** Fluoroscopic (30° right anterior oblique projection) catheter position during posterior fast pathway radiofrequency (RF) ablation. Radiofrequency application at this site resulted in ablation of the fast AV node pathway. Note that the position of the tip of the ablation catheter (ABL) is near the coronary sinus os, near the typical slow pathway location. **B,** Electrocardiographic leads III and aVF (continuous recording) before and after radiofrequency ablation. The PR interval prolonged from 136 to 268 ms. CS = coronary sinus catheter; HB = His bundle catheter; HRA = high right atrial catheter; RVA = right ventricular apex.

(Patient 4), retrograde atrial activity recorded by the His bundle catheter preceded that in the coronary sinus os by 10 ms. However, in this patient the His bundle potential could be recorded only from a catheter positioned slightly posterior to the coronary sinus os (Fig. 3A). Ablation directed immediately posterior to the "displaced" His bundle catheter abolished anterograde fast pathway conduction, resulting in prolongation of the AH interval from 78 to 220 ms. A presumed slow pathway potential was recorded posterior to the site of ablation (~2 cm posterior to the displaced His bundle catheter [Fig. 3B]). These data suggest that in contrast to the other patients with a posterior fast pathway exit site, this particular patient had posterior displacement of the entire AV node–His bundle apparatus.

Radiofrequency ablation. Application of radiofrequency energy (one to six applications, median of two) in the posteroseptal region in the first six patients resulted in permanent loss of anterograde fast pathway conduction and complete VA block (Table 1). Atrioventricular node reentrant tachycardia was noninducible, and AV node echo beats and dual AV node physiology were no longer present. Four of the six patients (nos. 1 to 4) had persistent first-degree AV block after posteriorly directed ablation. The AH interval increased from 70 ± 24 to 195 ± 35 ms, and the PR interval increased from 146 ± 27 to 270 ± 144 ms. In all four patients, transient (<60 s) complete AV block developed during application of radiofrequency energy, and in two of these patients, 2:1 AV block persisted for <24 h (patients 3 and 4) before 1:1 conduction resumed. After ablation, the anterograde AV node refractory period increased in three of the four patients from ≤150, 220 and 280 ms to 320, 420 and 450 ms, respectively, as did the AV node Wenckebach cycle length (from <300, 380 and 400 ms to 380, 460 and 570 ms, respectively), suggesting concurrent slow pathway damage.

Two patients had persistent high degree AV block after ablation. In patient 5, 2:1 AV node conduction developed after one radiofrequency application in the posteroseptal area. At

Table 1. Patient Characteristics and Electrophysiologic Results in Patients With a Posterior Fast Atrioventricular Node Pathway

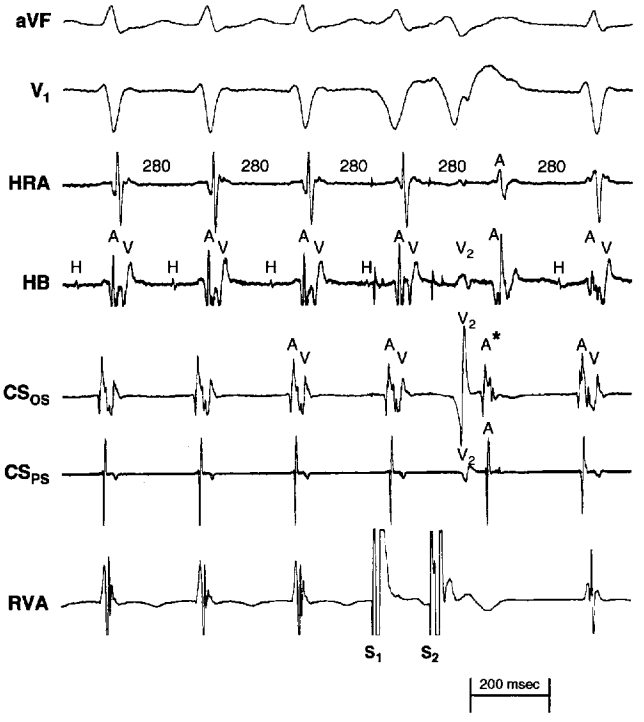
Pt No.	Age (yr)/ Gender	Cardiac Diagnosis	AVNRT (ms)				AVN Before/After RF Ablation (ms)					
			CL	AH	VA_{HB}	VA_{CSos}	AH	Ant WCL	Ant ERP Fast	Ant ERP Slow	Ret WCL	Ret ERP Fast*
1	19/M	Normal	230	170	0	0	45/160	< 300/380	250/†	≤ 150/320	< 300/no VA	≤ 190/no VA
2	67/F	Normal	395	345	20	20	100/170	n.a./320	520/†	≤ 210/≤ 180	n.a./no VA	n.a./no VA
3	16/F	Normal	280	225	20	0	56/230	380/460	400/†	220/420	< 260/no VA	≤ 210/no VA
4	67/F	Normal	320	270	0‡	10	78/220	400/570	370/†	280/450	360/no VA	≤ 210/no VA
5	47/M	Normal	360	320	0	0	120/2°AVB§	500/2°AVB	510/†	450/2°AVB	480/no VA	540/no VA
6	76/M	Normal	400	330	25	25	104/3°AVB	530/3°AVB	280/†	240/3°AVB	470/no VA	450/no VA
7	71/M	CAD	410	360	0	−2	97/95	400/390	480/350	350/†	400/390	340/340

*Dual retrograde pathways were not demonstrated in any patient (Pt). †Could not be determined because of ablation of this atrioventricular (AV) node pathway. ‡His bundle potential could be recorded only at a posteroseptal site slightly below coronary sinus os. §2:1 AV node conduction at sinus cycle length (CL) of 670 ms at end of ablation procedure; high grade AV block (AVB) with intermittent 1:1 conduction (PR interval of 560 ms) at 3 months. Ant = anterograde; AVN = AV node; AVNRT = AV node reentrant tachycardia; CAD = coronary artery disease; ERP = effective refractory period of the AV node; F = female; M = male; n.a. = could not be determined because of incessant AV node reentrant tachycardia before ablation; Ret = retrograde; RF = radiofrequency; VA = ventriculoatrial conduction; VA_{CSos} = VA interval measured at the coronary sinus os; VA_{HB} = VA interval measured in the His bundle recording; WCL = Wenckebach cycle length; 2°, 3° = second, third degree.

3 months there was variable AV node block with intermittent 1:1 AV conduction associated with a PR interval of 560 ms. Patient 6 developed persistent third-degree AV block after ablation, and a pacemaker was implanted. After a follow-up of 4 to 24 months, all patients were asymptomatic and were taking no antiarrhythmic medication.

The remaining patient (Patient 7) presented after analysis of the first six patients. Detailed mapping with the ablation catheter between the coronary sinus os and the His bundle confirmed that the earliest retrograde atrial activation occurred between the His bundle and the coronary sinus os (Fig. 4). To avoid fast pathway ablation, the ablation catheter was positioned slightly posterior to the earliest atrial activation site (fast pathway) during tachycardia, which corresponded to the site of a presumed slow pathway potential during sinus rhythm (Fig. 5). Radiofrequency energy was applied during atrial pacing in order to monitor the AH interval, and the power was gradually incremented from 10 to 30 W. Radiofrequency ablation at this site resulted in selective slow pathway ablation with preservation of anterograde and retrograde fast pathway conduction.

Figure 2. Posterior exit of the fast pathway during atrioventricular (AV) node reentrant tachycardia (Patient 3). Two ventricular extrastimuli are introduced during AV node reentrant tachycardia and dissociate ventricular activity from the tachycardia, clarifying the retrograde atrial activation sequence. Note that the earliest atrial activation (**asterisk**) is recorded 20 ms earlier in the coronary sinus os than in the His bundle (HB). Surface electrocardiographic leads aVF and V_1 are simultaneously shown with intracardiac recordings from the high atrium (HRA), His bundle, coronary sinus os (CS_{OS}), posteroseptal coronary sinus (CS_{PS}) and right ventricular apex (RVA). A = atrial activation; H = His bundle potential; V = ventricular activation; S = extrastimulus spike.



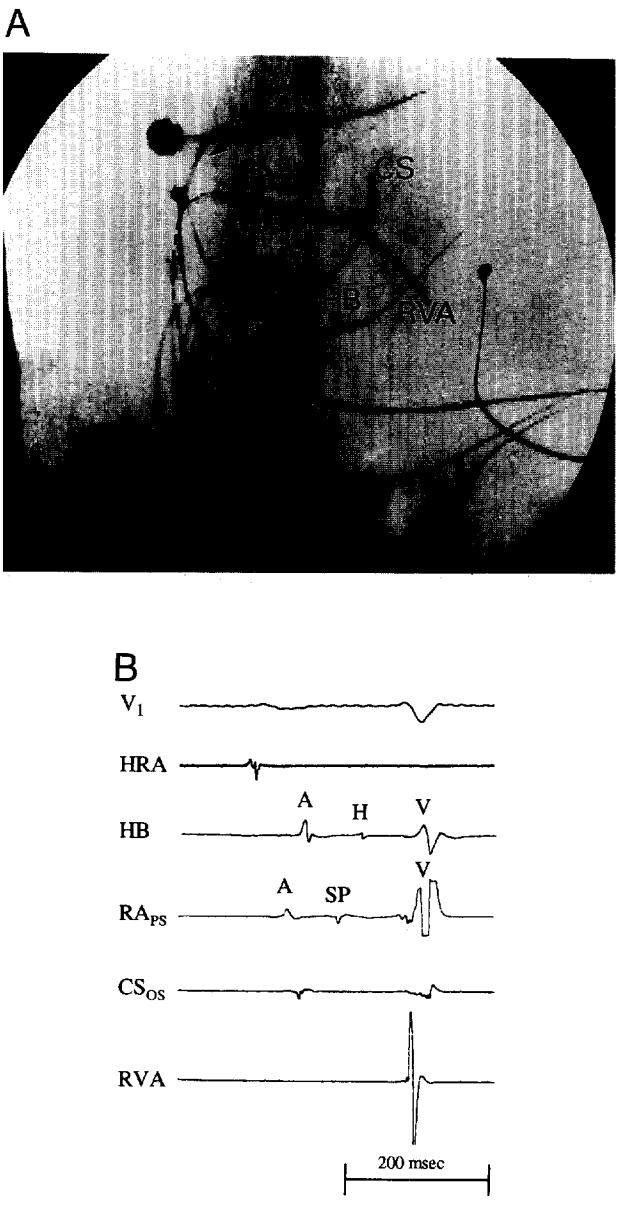


Figure 3. Posterior location of the AV node (Patient 4). **A,** Fluoroscopic (anteroposterior projection) location of the His bundle catheter (HB) located in the posteroseptal region. Ablation slightly posterior to this site resulted in fast pathway ablation. The position of the catheter labeled SP was 2 cm posterior to the site of fast pathway ablation and was at the recording site of a slow pathway (SP) potential. **B,** Sinus rhythm map showing a slow pathway potential recorded from the catheter position shown in **A.** Surface electrocardiographic lead V_1 is shown simultaneously with intracardiac recordings from the high atrium (HRA), His bundle (HB), posteroseptal right atrium (RA_{PS}), coronary sinus os (CS_{OS}) and right ventricular apex (RVA). Other abbreviations as in Figure 2.

Comparison of posterior and anterior pathways. To identify the characteristics of patients with a posterior fast pathway, we compared the clinical and electrophysiologic variables of the study group with those of a control group of 40 patients with AV node reentry and an anterior fast pathway. The results are summarized in Table 2. There were no significant differ-

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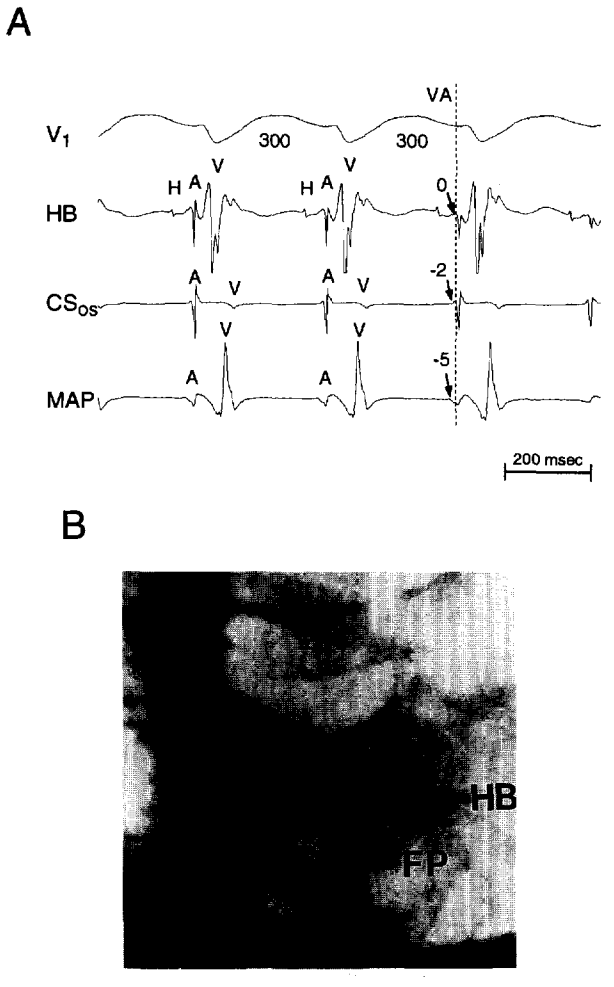

Figure 4. Posterior exit of the fast pathway (Patient 7). **A,** Activation map during AV node reentrant tachycardia with the ablation catheter recording from an intermediate site (MAP) between the His bundle catheter (HB) and the coronary sinus os (CS_{OS}). Note that earliest atrial activation is recorded earlier at the mapping site than at the coronary sinus os and the His bundle, confirming a posterior exit of the fast pathway. Surface electrocardiographic lead V_1 is simultaneously shown with intracardiac recordings from the His bundle (HB), coronary sinus os and intermediate mapping site (MAP). **B,** Right anterior oblique projection (30°) showing the catheter positions corresponding to the map in **A.** The mapping catheter is positioned at the site of the fast pathway (FP) exit. Other abbreviations as in Figure 2.

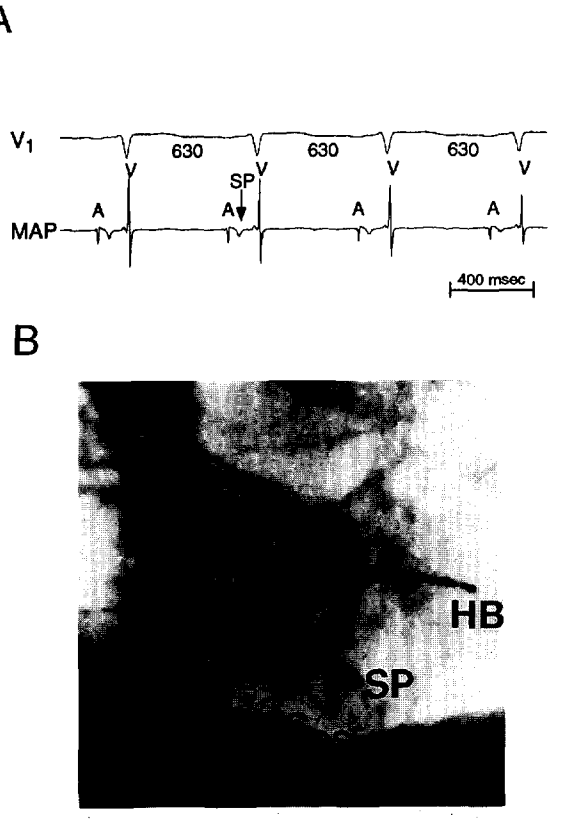

Figure 5. Selective slow pathway ablation in a patient with a posterior fast pathway (Patient 7). **A,** During sinus rhythm, a slow pathway potential (SP) was recorded with the mapping catheter (MAP) positioned at a site just below the fast pathway exit site (see Fig. 4). Radiofrequency ablation at this site resulted in selective slow pathway ablation. **B,** Right anterior oblique projection (30°) showing the catheter positions corresponding to panel **A.** The mapping catheter is positioned at the site of successful slow pathway (SP) ablation. Other abbreviations as in Figure 2.

ences in age, gender, cardiac diagnosis, baseline AV node conduction properties, cycle length and anterograde AV conduction during AV node reentrant tachycardia. In patients with a posterior fast pathway, atrial activation at the coronary sinus os clearly preceded atrial activation at the His bundle in only one patient. In five patients atrial activity at the two recording sites was nearly simultaneous during tachycardia. In the remaining patient, the entire AV node structure appeared posteriorly displaced, as described before. In contrast, simultaneous atrial activity during tachycardia at the His bundle and coronary sinus os was recorded in only 2 of the 40 patients with an anterior fast pathway. In these patients, posteroseptal radiofrequency ablation resulted in abolition of slow pathway conduction without an increment in the AH interval. In the remaining patients, atrial activity associated with the His bundle recording clearly preceded that recorded at the coronary sinus os during tachycardia, consistent with an anterior exit of the fast pathway.

Discussion

The findings of this study suggest that important exceptions exist to the emerging model of AV node reentry based on an anteriorly located fast pathway and a posterior slow pathway. Specifically, we identified patients with AV node reentry in whom a posterior fast AV node pathway comprised the retrograde limb of the reentrant circuit. These findings delineate the limitations of an anatomically guided ablation directed at the posterior slow AV node pathway because inadvertent fast pathway ablation may occur in patients with a posterior fast pathway. These results emphasize the importance of prospectively determining the difference between the

Table 2. Comparison of Patients With Posterior Fast Pathway and Anterior Fast Pathway

	Posterior Fast Pathway (n = 7)	Anterior Fast Pathway (n = 40)	p Value
Age (yr)	52 ± 25	50 ± 16	0.53
M/F (%)	57/43	33/67	0.21
Cardiac diagnosis (% normal)	86	72	0.89
AH interval (ms)	86 ± 27	86 ± 20	0.88
HV interval (ms)	49 ± 3	50 ± 7	0.97
Anterograde WCL (ms)	418 ± 84	361 ± 74	0.09
AVN ERP fast pathway: anterograde (ms)	399 ± 113	350 ± 106	0.27
AVN ERP slow pathway: anterograde (ms)	271 ± 100	258 ± 68	0.8
Retrograde WCL (ms)	385 ± 92	381 ± 103	0.76
AVN ERP fast pathway: retrograde (ms)	323 ± 146	282 ± 93	0.76
AVNRT			
CL (ms)	342 ± 68	348 ± 62	0.83
AH interval (ms)	289 ± 70	294 ± 64	0.83
HV interval (ms)	47 ± 6	50 ± 9	0.48
VA interval HB (ms)	9 ± 12	3 ± 18	0.34
VA interval CS_{OS} (ms)	7 ± 11	19 ± 19	0.07
$\Delta VA_{CSos} - VA_{HB}$ (ms)	−2 ± 9	16 ± 11	0.0003

Data presented are mean value (±SD), unless otherwise indicated. Abbreviations as in Table 1.

ventriculoatrial (VA) interval during tachycardia recorded at the coronary sinus ostium and His bundle catheter during slow–fast AV node reentry before radiofrequency ablation.

Atrionodal inputs. Experimental studies have demonstrated that rabbit and canine AV nodes have dual atrionodal inputs that are defined by anatomic obstacles to conduction rather than by specialized conducting tissue (18–20). These inputs consist of an anterior atrionodal input that runs anteriorly in the interatrial septum and a posterior input that travels by way of the crista terminalis and around the os of the coronary sinus in the posteroseptal region (20). Functionally distinct dual (anterior and posterior) atrial inputs have also been demonstrated in humans with and without AV node reentrant tachycardia (4). The observation that the slow AV node pathway can be ablated by application of radiofrequency energy in the right posteroseptal region of the tricuspid valve annulus (remote from the compact AV node) has prompted the suggestion that the atrionodal inputs may be important in the genesis of AV node reentrant tachycardia in humans and that the perinodal atrial tissue between the inputs may comprise a critical component of the reentrant circuit (1–4,8).

On the basis of these studies and ablative data from patients with AV node reentry (1–13), an anatomic and functional clinical model of an anterior fast AV node pathway and a posterior slow pathway has emerged. However, preliminary observations suggest that there may be important exceptions to this model. For example, the presence of a posterior fast or an anterior slow pathway has been inferred retrospectively after its unintentional ablation during attempts to ablate the reciprocal pathway (2,8,10,11). Reversal of fast and slow AV node pathways (anterior slow and posterior fast) has also been shown during intraoperative ice mapping (22).

Posterior fast pathways. Seven patients in this study had a posteriorly located fast pathway. Evidence supporting a posterior location of the fast pathway in these patients include 1) earliest retrograde atrial activation occurring between the His bundle and the coronary sinus os during slow–fast AV node reentrant tachycardia, and 2) anterograde and retrograde fast pathway ablation during application of radiofrequency energy in the tricuspid annulus posterior and inferior to the compact AV node (confirmed by prolongation of the AH interval and VA block). The fast pathway presumably traversed near the posterior slow pathway in these patients because transient or persistent high degree AV block was observed in all patients with posterior fast pathway ablation during the successful radiofrequency application. Damage to the slow pathway was incomplete and reversible because AV conduction resumed in five of six patients; however, there was evidence of residual slow pathway modification because the AV node Wenckebach cycle length and the anterograde refractory period of the slow pathway increased in these patients. Close physical proximity of the fast and slow AV node pathways was also demonstrated by detailed activation mapping in our last patient (Patient 7), in whom the slow pathway was selectively ablated just posterior to the fast pathway exit site (Fig. 4 and 5).

In all but one patient with a posterior fast pathway ablation, the ablation catheter was positioned closer to the coronary sinus os than to the His bundle catheter, at least 15 mm posterior to the His bundle catheter; no His bundle potential was observed at maximal amplification. In the remaining patient, the only area where a His bundle potential could be recorded was also at the inferoposterior aspect of the tricuspid valve annulus, at the level of the coronary sinus os (Fig. 3). The location of the ablation catheter was confirmed fluoroscopically immediately before, during and after ablation in all patients and remained stable during radiofrequency energy application. The number of radiofrequency applications for each patient was similar to that reported by others (2). Because pathologic studies have shown that radiofrequency energy delivered in a unipolar catheter configuration produces a well-demarcated lesion <10 mm in its greatest diameter (23), it is unlikely that the changes in AV node conduction noted in our study reflect inadvertent catheter movement or direct damage to a typically located compact AV node but rather resulted from ablation of an atypically located fast AV node pathway with preservation of slow pathway conduction. Similarly, high grade AV block in these patients most likely resulted from damage to an atypically located posterior fast AV nodal pathway as well as a typically located slow pathway. The present data do not permit us to determine whether or not this particular configuration represents an atypical location of the compact AV node.

The form of AV node reentry described previously (slow–fast with a posterior exit) is different from the tachycardia

reported in patients with posterior exit sites and HA intervals >110 ms (24,25). Although the AH interval is greater than the HA interval in these patients, thus excluding the fast-slow form of AV node reentry, this arrhythmia is consistent with AV node reentry as a result of an anterograde slow and a retrograde intermediate pathway with a posterior retrograde exit site (16). Identification of the posterior exit site in this form of AV node reentry is readily made (in contrast to the present study) because atrial activation is not obscured by nearly simultaneous atrial and ventricular activation.

In all patients but one, an optimal His bundle recording was obtained at the usual anteroseptal aspect of the tricuspid valve annulus. This finding suggests that the specialized conduction tissue was not displaced but that the observed variations in AV node pathway location may have been caused by anatomic variations in the atrionodal inputs. High resolution mapping of the perinodal region in humans with AV node reentrant tachycardia demonstrates higher conduction velocities in the anterior interatrial septum than in the posteroseptal region (26). The functional difference in conduction velocity between the anterior and posterior atrionodal inputs may result in part from discontinuous nonuniform anisotropic impulse propagation in perinodal atrial tissue (27), with conduction proceeding more rapidly along the longitudinal fiber axis than along the transverse axis. Aberration of fast or slow AV node pathway location could therefore result from changes in fiber orientation and associated functional changes in the preferential inputs into the AV node in selected patients. Because only one patient in this study had evidence for structural heart disease, it is likely that atypical locations of the fast AV node pathway represent a spectrum of normal anatomic variation.

Implications for radiofrequency ablation. Recognition of patients with a posterior fast AV node pathway may help avoid inadvertent heart block in patients undergoing radiofrequency ablation of AV node reentry. Mapping of the retrograde exit site during slow–fast AV node reentrant tachycardia is often obscured by simultaneous atrial and ventricular activation. Dissociation of ventricular activity from the tachycardia with ventricular extrastimuli enables clear distinction between the atrial and ventricular electrogram and facilitates mapping of the retrograde fast AV node pathway. However, despite this approach, the retrograde exit is still not always clearly delineated. Simultaneous atrial activation at the coronary sinus os and His bundle suggests an exit site intermediate to the two catheter positions and was a useful clue to the presence of a posterior fast pathway. As demonstrated in Patient 7, more detailed activation mapping in the posterior region may then precisely identify the location of the fast pathway and permit ablation of the slow pathway at a contiguous site with a slightly later retrograde atrial activation (corresponding to the site of a presumed slow pathway potential during sinus rhythm), thereby avoiding inadvertent fast pathway ablation. Furthermore, the importance of monitoring AV conduction properties during delivery of radiofrequency energy in these patients should be emphasized.

An interesting finding in this study is that transient complete AV block occurred during radiofrequency ablation, consistent with concurrent slow pathway damage, suggesting that transposition of the fast and slow pathways did not occur. These pathways thus by inference run in close proximity to each other and may account for the infrequent but well described examples of paradoxic heart block that complicate slow pathway ablation (10,11).

Perhaps most important, the anatomic variants of AV node pathways that these patients manifest, although relatively rare, highlight the limitations of a "blinded" posterior slow pathway approach. It is therefore essential to precisely map and localize not only the pathway targeted for ablation but also the reciprocal AV node pathway, because both pathways may course in close proximity to each other.

Study limitations. The presumed location of the fast AV node pathway is inferential and is based on the results of activation mapping and radiofrequency ablation. Mapping of the fast AV node pathway exit site was performed during retrograde conduction. However, the location of the anterograde fast AV node pathway may not be identical with that of the retrograde counterpart, which may explain why in some patients it is possible to ablate retrograde fast pathway conduction selectively without affecting fast anterograde conduction (1,9,28).

Conclusions. A model of an anterior fast and posterior slow AV node pathway is not universally applicable because there is evidence of a posteriorly located fast pathway in a subset of patients with AV node reentry. Recognition of posterior fast pathways by detailed mapping of retrograde atrial activation during tachycardia may avoid inadvertent collateral damage to the fast pathway during ablation of AV node reentrant tachycardia.

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